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Where the Eye Looks, the Hand Follows: Limb-Dependent Magnetic Misreaching in Optic Ataxia

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Summary

The posterior parietal cortex (PPC) is thought to play an important role in the sensorimotor transformations associated with reaching movements. In humans, damage to the PPC, particularly bilateral lesions, leads to impairments of visually guided reaching movements (optic ataxia). Recent accounts of optic ataxia based upon electrophysiological recordings in monkeys have proposed that this disorder arises because of a breakdown in the tuning fields of parietal neurons responsible for integrating *spatially congruent* retinal, eye, and hand position signals to produce coordinated eye and hand movements [1]. We present neurological evidence that forces a reconceptualization of this view. We report a detailed case study of a patient with a limb-dependent form of optic ataxia who can accurately reach with either hand to objects that he can foveate (thereby demonstrating coordinated eye-hand movements) but who cannot effectively *decouple* reach direction from gaze direction for movements executed using his right arm. The demonstration that our patient's misreaching is confined to movements executed using his right limb, and only for movements that are directed to nonfoveal targets, rules out explanations based upon simple perceptual or motor deficits but indicates an impairment in the ability to dissociate the eye and limb visuomotor systems when appropriate.

Results and Discussion

Optic ataxia (OA) refers to a disorder of visually guided reaching movements that is not attributable to a basic motor or sensory deficit [2–4]. The disorder was initially described by Reszö Bálint as one of a triad of visuospatial symptoms that can result from bilateral damage to the occipital-parietal cortex in humans, and which has since become known as Bálint's or Bálint-Holmes syndrome [5]. More recent studies have confirmed that optic ataxia can occur in isolation from the other symptoms associated with Bálint's syndrome and can also follow unilateral damage to the parietal cortex of either hemisphere (most frequently involving the intraparietal sulcus and the superior parietal lobule [SPL]) [6, 7].

Optic ataxia has most often been described as arising from a "disconnection" between visual processing systems and motor areas, and Bálint saw the disorder as arising largely as a consequence of the transection of white matter pathways running beneath the parietal cortices [2]. However, this disconnection viewpoint oversimplifies the important role played by parietal cortex in processing spatial information and in guiding eye and limb movements toward extrapersonal targets [8]. Recent electrophysiological studies in monkeys confirm this view insofar as they indicate that there is no single, supramodal map of space that is used to guide movements. Instead, movements appear to be capable of being planned and controlled within multiple coordinate systems, each one attached to a different body part [9–12]. For example, electrophysiological studies of the SPL have demonstrated the existence of a "parietal reach region" in which the locations of the targets for *reaching* movements are coded in *eye-centered* coordinates [13].

Based largely upon electrophysiological recording studies in nonhuman primates, recent theoretical accounts of optic ataxia have attempted to reframe the disconnection account as a breakdown in the tuning fields of parietal neurons responsible for producing coordinated eye-hand movements [1]. Within this view, neurons within the SPL are thought to integrate spatially congruent retinal, eye, and hand position signals to produce coordinated eye and hand movements [1]. Importantly, it is proposed that the role of these global tuning fields is to combine eye and hand signals that code for the same direction as follows: "…signals encoded and combined within the global tuning field of each individual cell share a common property: they all point in the same direction. In this context, it is worth stressing that combination of retinal, eye and hand signals is regarded as a necessary prerequisite of reaching to visual targets by virtually all coding hypotheses …" (p. 233).

Here we present detailed evidence in support of the view that the most common form of optic ataxia, so-called nonfoveal optic ataxia [14], may result less from a failure to produce spatially congruent and coordinated eye and hand movements as from a failure to *decouple* the eye and limb visuomotor systems so that each might undertake simultaneously independent actions.

We examined visually guided reaching movements executed by a 65-year-old right-handed male (JJ) who presents with Bálint's Syndrome following recurrent cerebral hemorrhages over a period of 6 years. A recent MRI scan of JJ (Figure 1) reveals asymmetrical bilateral posterior atrophy predominantly of the parietal lobes, with the damage to the left parietal cortex extending farther into the superior region than in the right hemisphere. Testing with finger presentation and Goldmann perimetry revealed an absolute visual field deficit in the

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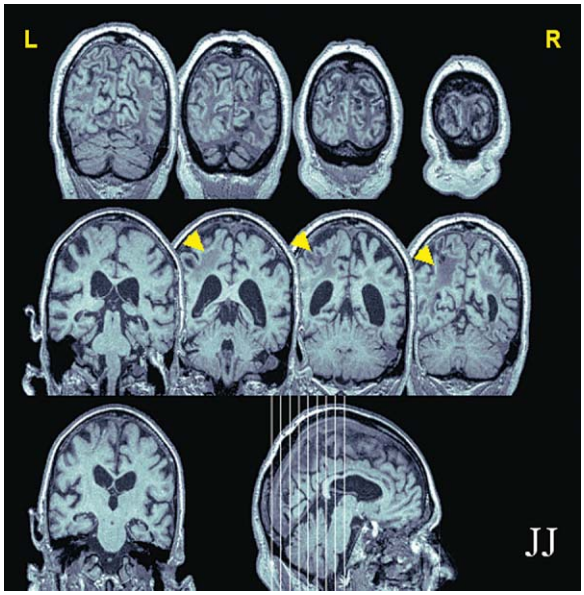

Figure 1. JJ Structural MRI Scan

T1-weighted MRI scan showing asymmetrical bilateral atrophy predominantly of the parietal lobes with the damage to the left parietal cortex extending farther into the superior region than is the case within the right hemisphere. Damage to the left hemisphere involved the SPL and IPS and the angular gyrus. In addition, there was signal change consistent with degeneration of the white matter in the occipital lobe. Damage to the right hemisphere involved the IPS and the posterior aspect of the angular gyrus. The yellow arrowheads indicate areas of asymmetric signal in the left hemisphere.

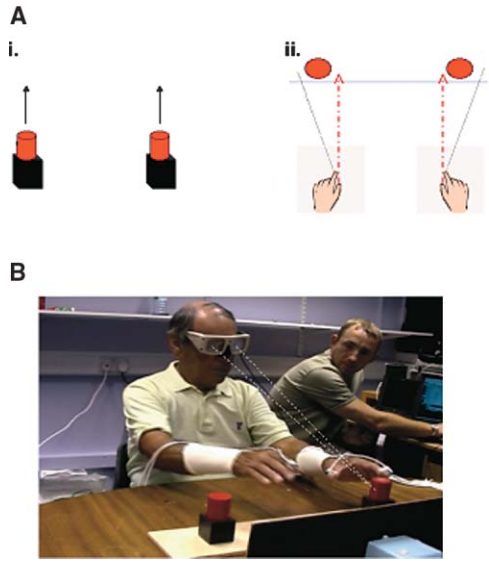

Figure 2. Task Characteristics

(A) Illustration of the bimanual reaching task. (i) JJ was instructed to reach unimanually or bimanually to grasp one or both of two red wooden dowels presented shoulder width apart. (ii) During reaching movements, the spatiotemporal pattern of the movements of each of JJ's thumbs was tracked using an electromagnetic tracking device sampling at 81 Hz.

(B) Shows a single frame of video taken while JJ was executing a bimanual reaching movement. In this trial, JJ was instructed to look at the left target object (see text for details). Note that as JJ's left hand approaches the left target object, his right hand is substantially leftward of the right target object.

left inferior quadrant. JJ shows no clinical signs of visuospatial neglect and his somatosensory function is normal (see [16, 17] for further details). Unimanual and bimanual reach-to-grasp movements to visually defined targets were examined in JJ using a task that we have utilized previously to examine reaching movements in brain-injured individuals [18, 19] (Figure 2).

Analyses of end-point errors demonstrated that JJ could accurately execute unimanual reach-to-grasp movements using his right and left hands if he was permitted to foveate the target object (Figure 3A; comparison with data from healthy control subjects revealed that JJ's end-point errors were within one SD of controls). Furthermore, informal testing with JJ on several occasions verified that he could also accurately execute bimanual reach-to-grasp movements using both hands if the movement was made *without vision* (i.e., blindfolded) and to the remembered locations of target objects, thus confirming previous reports that non-visually guided movements can be performed accurately [2, 16, 20]. In contrast, while JJ's left hand remained as accurate during bimanual reach-to-grasp movements as was the case for unimanual movements using his left hand [$F(1,19) = 1.5$, $p > 0.2$], this was not so for his right hand, which exhibited substantial leftward errors during bimanual movements [Figure 3B; $F(1,19) = 250.9$, $p < 0.0001$].

To investigate the cause of these errors, we repeated this basic experimental task in several different ways. First, we had JJ execute bimanual reaches while fixating either the rightward or the leftward target object. Analyses of end-point errors under these conditions revealed that JJ could accurately reach with *both* arms while fixating the rightmost target (Figure 3C), but he was completely unable to execute accurate reach-to-grasp movements with his right arm when fixating the leftmost target, producing large leftward errors on all trials (Figure 3D). Statistical analyses confirmed that gaze angle had no effect on reaching movements executed with the left arm [$F(2,18) < 1.0$, $p = 0.4$], but gazing at the left target led to significant leftward errors for reaches of the right arm [Figure 4, left; $F(2,18) = 26.6$, $p < 0.0001$]. This finding confirms previous brief reports of so-called magnetic misreaching in which reaching movements appear to be locked to the object at fixation [14, 15, 21]. Second, to test that this effect was not due to visual capture by the limb reaching toward the leftmost objects, we had JJ perform the above task under two forms of open-loop control. In one, he was instructed to foveate either the rightmost or leftmost target object, and vision to both eyes was occluded (using a set of LCD lenses) at movement onset (Figure 2B shows JJ wearing the LCD spectacles used). In another, JJ reached to the "virtual" locations of visual targets with his limbs occluded from view. In both cases, JJ exhibited accurate movements of both arms while looking rightwards [$F(1,29) < 1.0$, $p > 0.1$] but again demonstrated profound reaching errors when asked to fixate the left target [Figure 4, right; $F(1,29) = 94.7$, $p < 0.0001$]. Third, to test that JJ's misreaching was not the result of a

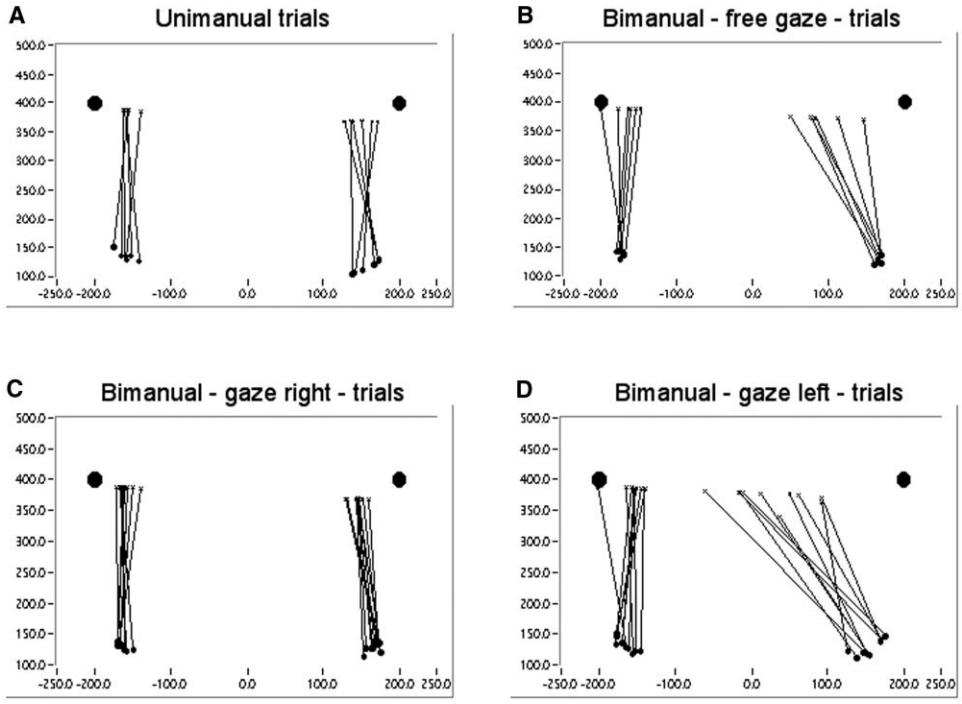

Figure 3. Movement Accuracy

Movement vectors indicating the direction of JJ's right and left thumbs on individual trials during (A) unimanual reaching trials, (B) bimanual reaching trials, (C) bimanual reaching trials in which JJ was required to fixate the right target object, and (D) bimanual reaching trials in which JJ fixated the left target object. Note that in all cases, accurate reaching movements will result in the thumb slightly to the left or right (depending upon the hand used) of the object's center. Also, JJ's end-point errors on unimanual trials were within one standard deviation of healthy control subjects.

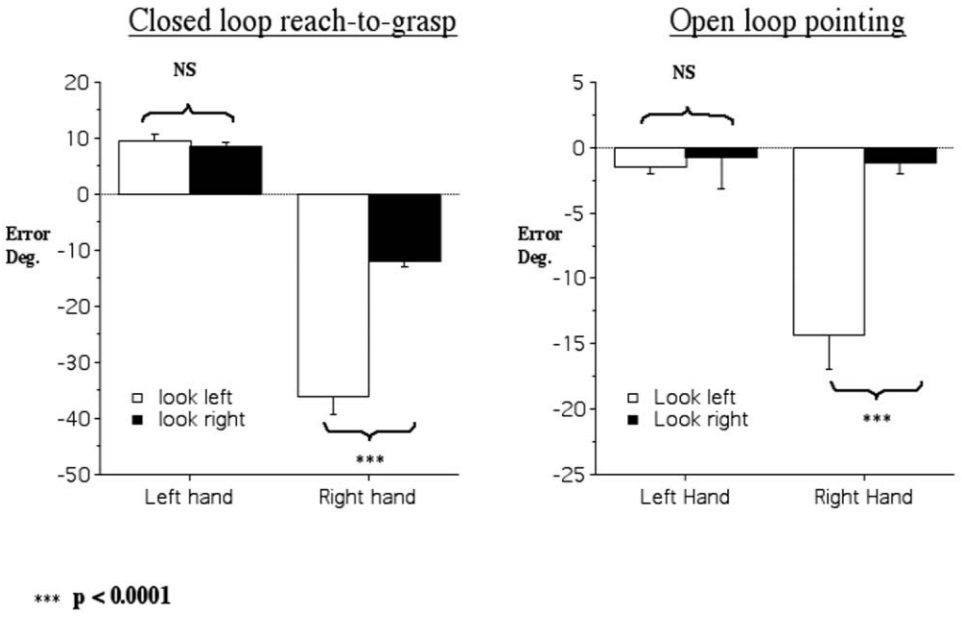

Figure 4. Mean Direction Errors for Patient JJ while Executing Bimanual Reaching Movements to Two Targets while Fixating the Left or Right Target

Negative values indicate a leftward error. The left panel shows reach-to-grasp movements made with full visual feedback of the hand and target throughout the reach. The right panel shows data for pointing movements executed beneath an opaque surface in which JJ had no visual feedback of his limb but could see the target object throughout the reach. In both cases, JJ made large leftward direction errors with his right hand, but only when he was required to fixate the left target object. Gaze direction had no effect on reaching movements executed by JJ using his left hand. Error bars are standard errors.

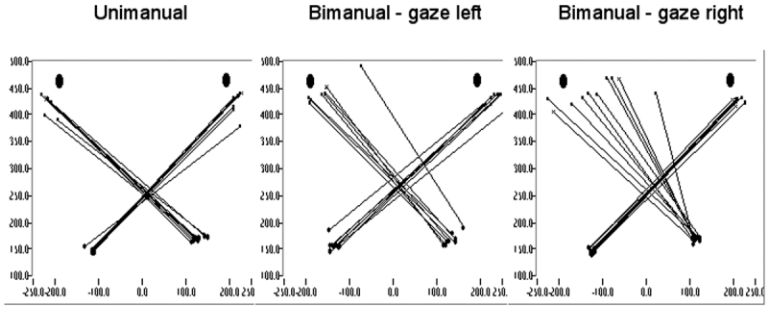

Figure 5. Crossed Arms Condition

Movement vectors indicating the movement direction of JJ's right and left thumbs during unimanual (left), bimanual gaze-left (center), and bimanual gaze-right (right) reach-to-grasp movements.

"field" effect, in which misreaching errors are simply biased toward one side of peripersonal space, we ran a "crossed" hands condition in which JJ reached for left targets with his right hand and right targets with his left hand. If JJ's misreaching errors are due to a leftward field effect, then he should continue to produce leftward errors in the crossed hands condition. However, if JJ's misreaching errors are due to a failure to decouple his right arm from where he is looking, then the direction of JJ's misreaching errors should reverse direction, i.e., he should now produce rightward errors when reaching with his right arm. The results of this experiment are shown in Figure 5. This figure reveals that JJ can accurately execute crossed unimanual movements with both hands and that movements of JJ's left hand remain accurate during crossed bimanual movements [maximum $F(1,9) < 1.0$, $p > 1.0$]. In contrast, JJ's right hand exhibits large *rightward* errors during crossed bimanual movements only when he was required to fixate the rightmost object [$F(1,9) = 26.7$, $p < 0.001$]. Finally, to establish that JJ's misreaching during bimanual movements was not simply a consequence of his more impaired right hand following his left hand or due to joint attention to a single location, we had JJ execute unimanual reaching movements toward foveally or extrafoveally presented targets. In this study, JJ fixated at one of two locations and targets were presented at either the fixated (foveal) or nonfixated (extrafoveal) locations. Reaches were made unimanually with right and left arms. Mean reaching errors are presented in Figure 6. Inspection of Figure 6 shows that JJ was not impaired at reaching to extrafoveal targets with his left arm [extrafoveal $= -0.928°$ versus foveal $= -2.224°$; $F(1,9) < 1.0$, $p = 0.5$]. In contrast, unimanual reaching movements executed using his right arm showed a significant increase in end-point errors—toward the point of fixation—when directed to nonfoveal targets [extrafoveal $= 8.818°$ versus foveal $= 1.998°$; $F(1,9) = 14.3$, $p < 0.005$]. These data confirm that JJ's limb-dependent magnetic misreaching extends to unimanual reaching to extrafoveal targets.

The data presented above confirm that patient JJ presents with a limb-dependent form of nonfoveal optic ataxia and has a number of implications for how we view this disorder. First, the demonstration that JJ can accurately execute reaching movements with his right hand during unimanual reaches and to bimanual reaches where he is permitted to foveate the target for his right hand demonstrates that his misreaching errors cannot be attributed to a basic motor deficit. Second, in contrast to previous reports (e.g., [14, 15, 21]), JJ's magnetic misreaching effect is confined to movements executed using his right limb that are directed to nonfoveal targets. Furthermore, the direction of JJ's misreaching errors reverses when he is required to reach for leftward targets using his right hand in the crossed reaching condition. Together, these observations rule out any explanation in terms of a simple perceptual deficit. Third, as noted previously [14], the fact that JJ can accurately reach for objects that he can foveate but produces large and systematic errors when reaching for nonfoveal targets also effectively rules out any simple disconnection account of OA. Finally, and most importantly, the pattern of misreaching deficits observed in patient JJ cannot be readily accounted for by a breakdown in mechanisms

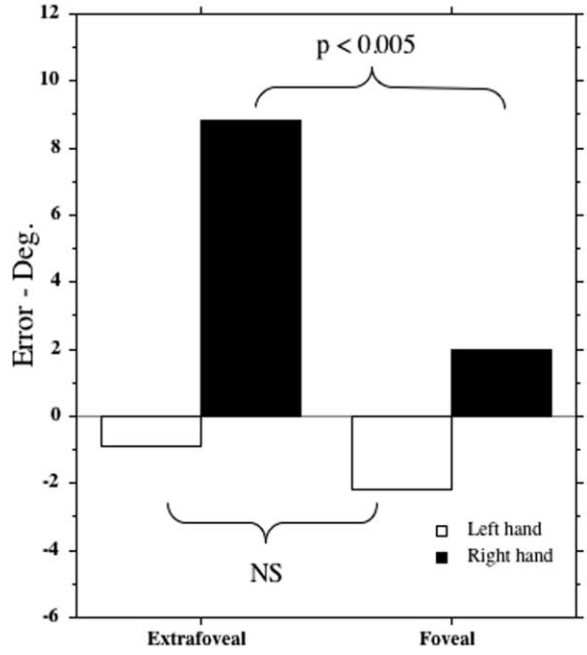

Figure 6. Mean Direction Errors for Patient JJ while Executing Unimanual Reaching Movements Using His Right or Left Upper Limb Were Directed to the Same Target Location that Was Viewed Foveally or Extrafoveally

The data illustrate that the accuracy of movements executed by JJ using his left hand were not significantly different when the target was viewed foveally or extrafoveally [$F(1,9) < 1.0$, $p = 0.5$]. In contrast, reaches executed using his right hand to extrafoveal targets were significantly less accurate than reaches executed to the same location when viewed foveally [$F(1,9) = 14.3$, $p < 0.005$].

that are responsible for producing coordinated eye-hand movements that combine eye and hand signals that code for the same direction. As has been demonstrated here, JJ has absolutely no problem in producing accurate reaching movements, with either arm, to objects that he can foveate (i.e., coordinated eye and hand movements that are spatially congruent). In contrast, he has severe difficulties in executing movements with his right arm that must be executed to target objects that lie outside his current point of gaze. Thus, JJ's misreaching errors arise less from a failure of mechanisms responsible for producing coordinated eye and hand movements as from a breakdown in the sensorimotor transformation processes that permit reach direction and gaze direction to be flexibly decoupled from one another so that they may undertake independent actions. A key aspect of this mechanism may be the ability to rapidly and efficiently switch between multiple coordinate systems (e.g., eye-centered or postural) according to task demands [14, 15]. The functional asymmetry observed for JJ's reaching movements could arise as a consequence of the asymmetry of the damage to left and right parietal cortices observed in JJ (i.e., damage is both more anterior and superior on the left than on the right). Alternatively, JJ's behavioral asymmetry could have arisen because of a hemispheric asymmetry in function in which the right hemisphere may be biased toward coding visual targets for reaching movements in extrinsic (e.g., eye-centered) coordinates and the left hemisphere may be similarly biased to code targets in intrinsic (e.g., postural or muscle) coordinates. Currently, our data do not distinguish between these alternatives.

In summary, several lines of evidence point to the important role played by the posterior parietal cortex (PPC) in carrying out the sensorimotor transformations that underlie visually guided reaching. In humans, lesions of the PPC, particularly those involving the intraparietal sulcus, lead to misreaching to visual targets—optic ataxia. Recent theoretical accounts of optic ataxia, based largely on single-unit electrophysiology in monkeys, propose that it arises as a result of a breakdown in the tuning fields of parietal neurons responsible for producing coordinated eye-hand movements [1]. Here, we have presented neurological evidence that the most common form of optic ataxia—nonfoveal optic ataxia—cannot be readily explained by this account. Specifically, we demonstrate that a key aspect of this form of optic ataxia may be less a breakdown in mechanism producing coordinated eye/hand movements so much as a failure to successfully decouple the eye and limb visuomotor systems so that each might undertake simultaneously independent actions.

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References

1. Battaglia-Mayer, A., and Caminiti, R. (2002). Optic ataxia as a result of the breakdown of the global tuning fields of parietal neurones. Brain *125*, 225–237.
2. Bálint, R. (1909). Seelenlähmung des 'schauens', optische ataxie, räumliche störung der aufmerksamkeit. Monattsschrifte für Psychiatrische Neurologie *25*, 51–81.
3. Harvey, M., Milner, A.D. (1995). Translation of R. Balint's Psychic paralysis of gaze, optic ataxia, and spatial disorder of attention. Cogn. Neuropsychol. *12*, 261–281.
4. Husain, M., and Stein, J. (1988). Rezsö Bálint and his most celebrated case. Neurology *45*, 89–93.
5. Holmes, G. (1918). Disturbance of visual orientation. Brit. J. Opthalmol. *2*, 449–468.
6. Ratcliffe, G., and Davies-Jones, G.A.B. (1972). Defective visual localisation in focal brain wounds. Brain *95*, 49–60.
7. Perenin, M.T., and Vighetto, A. (1988). Optic ataxia: a specific disruption in visuomotor mechanisms. Different aspects of the deficit in reaching for objects. Brain *111*, 643–674.
8. De Renzi, E. (1996). Balint-Holmes syndrome. In Classic Cases in Neuropsychology, C. Code, C.-W. Wallesch, Y. Joanette, and A. Roch Lecours, eds. (Hove, UK: Psychology Press), pp. 123–143.
9. Gross, C.G., and Graziano, M.S.A. (1995). Multiple representations of space in the brain. Neuroscientist *1*, 43–50.
10. Andersen, R.A., Snyder, L.H., Bradley, D.C., and Xing, J. (1997). Multimodal representation of space in the posterior parietal cortex and its use in planning movements. Annu. Rev. Neurosci. *20*, 303–330.
11. Rizzolatti, G., Fogassi, L., and Gallese, V. (1997). Parietal cortex: from sight to action. Curr. Opin. Neurobiol. *7*, 562–567.
12. Snyder, L.H. (2000). Coordinate transformations for eye and arm movements in the brain. Curr. Opin. Neurobiol. *10*, 747–754.
13. Batista, A.P., Buneo, C.A., Snyder, L.H., and Andersen, R.A. (1999). Reach plans in eye-centred coordinates. Science *285*, 257–260.
14. Buxbaum, L.J., and Coslett, H.B. (1997). Subtypes of optic ataxia: reframing the disconnection account. Neurocase *3*, 159–166.
15. Buxbaum, L.J., and Coslett, H.B. (1998). Spatio-motor representations in reaching: evidence for subtypes of optic ataxia. Cogn. Neuropsychol. *15*, 279–312.
16. Jackson, S.R., Newport, R., Mort, D., Husain, M., Jackson, G.M., Swainson, R., Pears, S., and Wilson, B. (2005). Action binding and the parietal lobes: some new perspectives on optic ataxia. In Attention in Action: Advances from Cognitive Neuroscience, G.W. Humphreys and M.J. Riddoch, eds. (Hove, UK: Psychology Press).
17. Jackson, G.M., Swainson, R., Mort, D., Husain, M., and Jackson, S.R. (2004). Implicit processing of global information in Balint's Syndrome. Cortex *40*, 177–178.
18. Jackson, G.M., Jackson, S.R., Husain, M., Harvey, M., Kramer, T., and Dow, L. (2000). The coordination of bimanual prehension movements in a centrally deafferented patient. Brain *123*, 380–393.
19. Jackson, G.M., Jackson, S.R., and Hindle, J.V. (2000). The control of bimanual reach-to-grasp movements in hemiparkinsonian patients. Exp. Brain Res. *132*, 390–398.
20. Milner, A.D., Dijkerman, H.C., Pisella, L., McIntosh, R.D., Tilikete, C., Vighetto, A., and Rossetti, Y. (2001). Grasping the past: delay can improve visuomotor performance. Curr. Biol. *11*, 1896–1901.
21. Carey, D.P., Coleman, R.J., and Della Sala, S. (1997). Magnetic misreaching. Cortex *33*, 639–652.